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MRI correlates of vascular cerebral lesions and cognitive impairment

Summary

The incidence of dementia is increasing to the stunning number of 30.000 new cases each year in the Netherlands. Despite progress in knowledge of the several forms of dementia, causal therapies are yet not available at this moment. However, several therapeutic agents are being investigated in different groups of dementia and it is therefore important to be able to differentiate between different causes of dementia. This especially holds for the distinction between Alzheimer's disease versus vascular dementia.

In the past, brain imaging was regarded as optional, mainly to exclude possibly surgically treatable causes of dementia, such as mass lesions. Today imaging can contribute to specifically diagnose the illness underlying a dementia syndrome, for example by demonstrating medial temporal lobe atrophy in Alzheimer's disease. MRI is very sensitive to vascular lesions. With T_1 -weighted series, cortical infarcts and lacunes can be visualized. T_2 -weighted sequences, like Fluid-Attenuated Inversion Recovery (FLAIR) and dual-echo Turbo Spin Echo (TSE) are used to show subcortical lesions; FLAIR is able to distinguish between WMH on one hand and lacunes and perivascular spaces on the other, while TSE is preferred for the basal ganglia (and thalamus) and infratentorial regions.

VaD is thought to be the second most common type of dementia. The NINDS-AIREN criteria for VaD are the most recent and widely used. These criteria contain a radiological part, necessitating evidence of vascular disease on brain imaging. In chapter 2.2 we showed that overall interobserver agreement of these radiological criteria is poor (Cohen's $\kappa = 0.29$), and that operationalization only improves agreement between already experienced raters of vascular lesions on MRI ($\kappa = 0.62$), but that it does not affect agreement between raters who are not trained in scoring vascular lesions (from $\kappa = 0.17$ to $\kappa = 0.18$ after operationalization).

According to the NINDS-AIREN radiological criteria for VaD, bilateral vascular lesions in the thalamus can account for a VaD syndrome. In Chapter 2.3, axial TSE imaging is compared to axial FLAIR with respect to these thalamic lesions. In a blinded review of MRIs of 73 subjects, meeting the NINDS-AIREN radiological criteria of VaD, TSE was found to be more sensitive. Using FLAIR, only 55% of the probable vascular lesions are detected. For TSE, this is 97%. Those results signify that, when applying the NINDS-AIREN criteria, FLAIR should not be the only T_2 -weighted sequence.

WMH can be assessed qualitatively, using a visual scale, or quantitatively, with volumetric methods. Chapter 3.1 shows the characteristics of several visual scales for WMH in comparison with a semiautomated volumetric assessment. The visual scales appear to have ceiling effects and their relationship with increase in WMH volume is not linear, possibly compromising their ability to detect differences between groups. Several visual scales for the assessment of WMH were investigated with respect to their power of detecting WMH changes over time in chapter 3.2. It is reported that on 3-year interval MRIs of subjects with varying degree of WMH, the rating scales that are developed for the assessment of WMH on cross-sectionally, are largely unable to pick up changes in WMH burden over time within subjects. Volumetric assessment or the use of a visual rating scale, specifically designed to detect change over time, are more sensitive to progression of WMH, and are therefore preferred in longitudinal studies.

Vascular lesions can lead to VaD, but there is a growing body of evidence that they also might play a role in the etiology of AD. Vascular lesions are more common in subjects with AD compared to healthy controls of the same age. We investigated the presence of WMH in a sample of subjects with amnesic MCI, which is regarded by some as a transition phase between normal aging and AD, and who were followed up over a period of three years. Using a visual rating scale, WMH was assessed in several brain regions: subcortical, periventricular, in the basal ganglia, and infratentorial. We found that WMH located in the periventricular regions was significantly more prevalent in the individuals who later progressed to AD. This may indicate that vascular factors influence the development of degenerative dementia such as AD.

Ischemic vascular brain lesions can be divided into cortical (large vessel) infarcts, lacunar (subcortical) infarcts and WMH (subtotal ischemia). In chapter 4.2 we investigated risk factor profiles of these three types of CVD and found that male gender is a risk factor for all of these CVD types.